DISSERTATION ON

STUDY ON PROGNOSTIC VALUE OF ELECTROPHYSIOLOGICAL TESTS AND EFFICACY OF STEROIDS AND ACYCLOVIR IN BELL'S PALSY

Submitted in partial fulfilment of
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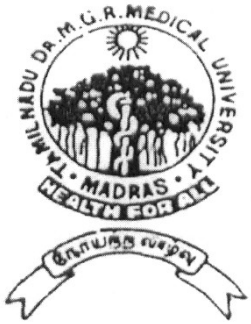

MADRAS MEDICAL COLLEGE
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CERTIFICATE

This is to certify that this dissertation entitled "**STUDY ON PROGNOSTIC VALUE OF ELECTROPHYSIOLOGICAL TESTS AND EFFICACY OF STEROIDS AND ACYCLOVIR IN BELL'S PALSY**" submitted by **Dr. Y.KINGSLY JEBASINGH appearing** for **D.M.,** Degree examination in **August 2007**, is a bonafide record of work done by him under my direct guidance and supervision, in partial fulfillment of regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai. I forward this to the Tamil Nadu Dr. M.G.R. Medical University, Chennai, Tamil Nadu, and India.

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DECLARATION

I solemnly declare that the dissertation titled "**STUDY ON PROGNOSTIC VALUE OF ELECTROPHYSIOLOGICAL TESTS AND EFFICACY OF STEROIDS AND ACYCLOVIR IN BELL'S PALSY "** is done by me at the Institute Of Neurology, Madras Medical College & Govt. General Hospital, Chennai during 2005-2007 under the guidance and supervision of Prof**. V. Natarajan.**

The dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University towards the partial fulfillment of requirements for the award of D.M., in Neurology.

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CONTENTS

INTRODUCTION

INTRODUCTION

Bell's palsy is the most frequent disease of the seventh cranial nerve, with a good prognosis. More than 70% patients attain complete clinical recovery, with no noticeable residua [1]. Persistent sequellaes are usually noted in cases with profound axonal loss only. Electrophysiological tests may offer valuable information in defining the severity of nerve injury and a possible residual dysfunction [2]. Treatment of Bell's palsy is still controversial. Therapy is difficult to evaluate, because as many as two thirds of patients with Bell's palsy spontaneously recover and achieve near-normal function. Many patients begin to improve as early as 10 days after the onset, even without treatment [3]. Drug therapy mainly consists of corticosteroids with or without an antiviral (acyclovir). These drugs hasten the recovery and lessen the ultimate degree of dysfunction.

One hundred and one patients with clinical signs of Bell's palsy were included in this study. Clinical signs were recorded (the degree of facial muscle palsy) and treated with steroids and acyclovir in combination or separately. Electrophysiological tests (amplitude and latency of compound muscle action potential CMAP) were done on first week and fourteenth day of the onset of palsy. The results were analysed in regard to the usefulness of electrophysiological studies in prognostication and which combination of drugs is most useful in management of Bells palsy.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Historical review

As early as 1821, Sir Charles Bell discovered that the facial nerve is responsible for facial muscle movement (Bell, 1821). Soon thereafter, the recognition of the anatomic basis of facial nerve injury gave way to reconstructive strategies. The first facial nerve repair was performed in 1879 by Drobnick Bunnell. He attempted an intratemporal repair of the facial nerve in its fallopian canal course.

In 1971, Thompson became one of the pioneers in free muscle transplantation without vascular microanastomosis. He discovered that the best results were achieved when the graft had been denervated 2-3 weeks before transplantation. In 1973, May and Miehlke successfully conducted the fascicular spatial orientation (motor, secretory, afferent) of the facial nerve. Scaramella pioneered cross-facial nerve grafts as a technique of coapting contralateral intact facial nerves to injured facial nerve.

Harii et al broached microneurovascular free muscle transplantation for the reconstruction of the paralyzed face in 1976. This group performed a free gracilis muscle transfer for the reconstruction of a smile with excellent results.

Facial Nerve- Anatomic Considerations

The seventh cranial nerve is mainly a motor nerve supplying all the muscles concerned with facial expression on one side. The sensory component is small (the nervus intermedius of Wrisberg). It conveys taste sensation from the anterior two – thirds of the tongue and, variably, cutaneous sensation from the anterior wall of the external auditory canal. The taste fibers first traverse the lingual nerve (a branch of the trigeminal) and then join the chorda tympani, which convey taste sensation via the facial nerve to the nucleus of the tractus solitarius. Secretomotor fibers innervate the lacrimal gland through the greater superficial petrosal nerve and the sublingual and submaxillary glands through the chorda tympani.

Several other anatomic facts are worth remembering. The motor nucleus of the seventh nerve lies ventral and lateral to the abducens nucleus, and the intrapontine fibers of the facial nerve partly encircle and pass ventrolaterally to the abducens nucleus before emerging from the pons, just lateral to the corticospinal tract. At their juxtaposition in the floor of the upper fourth ventricle, the sixth and seventh nerves may be affected simultaneously by a vascular or infiltrative lesion.

The facial nerve enters the internal auditory meatus with the acoustic nerve and then bends sharply forward and downward around the anterior boundary of the inner ear. At this angle (genu) lies the sensory ganglion (named geniculate because of its proximity to the genu). The nerve continues in its own bony channel, the facial canal, within which. 1st distal to the geniculate ganglion, it provides a, branch to the pterygopalatine ganglion, i.e., the greater superficial petrosal nerve; somewhat more distally, it gives off a small branch to the stapedius muscle and is joined by the chorda tympani. It makes its exit from the skull at the stylomastoid foramen, then passes through the parotid gland and subdivides into five branches that supply the facial muscles, the stylomastoid muscle, the platysma, and the posterior belly of the digastric muscle.

A complete interruption of the facial nerve at the stylomastoid foramen paralyzes all muscles of facial expression. The corner of the mouth droops, the creases and skin folds are effaced, the fore head is unfurrowed, the palpebral fissure is widened, and the eye lids will not close. Upon attempted closure of the lids, both eyes roll upward (Bell's phenomenon), but the one on the paralyzed side remains visible. The lower lid sags also, and the punctum falls away from the conjunctiva, permitting tears to spill over the cheek. Food collects between the teeth and cheek, and saliva may dribble from the corner of the mouth. The patient complains of a heaviness or numbness and sometimes an aching pain in the face, but sensory loss can usually not be demonstrated. Taste, however, is intact because the lesion is beyond the site where the chorda tympani has separated from the

main trunk of the facial nerve.

If the lesion is in the facial canal above the junction with the chorda tympani but below the geniculate ganglion, all the preceding symptoms occur. In addition, taste is lost over the anterior two- thirds of the tongue on the same side. If the nerve to the stapedius muscle is involved, there is hyperacusis (painful sensitivity to loud sounds). With a stethoscope in the patient's ears, a tuning fork at the bell is louder on the side of the paralyzed stapedius muscle. If the geniculate ganglion or the motor root proximal to it is involved, lacrimation and salivation may be reduced. Lesions at this point may also affect the adjacent eighth nerve, causing deafness, tinnitus, or dizziness

Bell's palsy

Bell-s palsy is an idiopathic peripheral disease of the seventh cranial nerve. This is the most frequent cranial mononeuropathy with an annual incidence of 10 to 40 cases per 100,000 population with geographical variations. [1, 4] It can occur at any age, but mostly in the third and fourth decade of life. The disease was described as a distinct entity by Sir Charles Bell in 1893, and since then it has commonly been referred to as Bell's palsy. It is seen as often in men as in women. Those at high risk include pregnant women and people with diabetes mellitus. About 10% of those with Bell's palsy have a family history of the condition.

Each facial nerve travels through a narrow bony canal in the skull beneath the ear to the muscles on each side of the face. Acute inflammation and edema of the facial nerve are thought to lead to entrapment of the nerve in the bony canal, which leads to compression ischemia. An observation that supports the hypothesis of an inflammatory etiology is that the facial nerve shows enhancement on magnetic resonance imaging (MRI) in patients with acute Bell palsy. [5]

Etiology and role of herpes simplex virus

Events, such as viral infection [6, 7, 8], ischemia [9], and autoimmune reaction [10], have been proposed as causes of Bell's palsy. Viral infection is thought to be the most likely cause [11]. However, it is rare to find a diagnostic fourfold increase in specific viral antibody titer in the acute and convalescent serum specimens of patients with Bell palsy [7, 8,]. Postmortem histopathologic studies of the facial nerve suggest viral neuritis [12], but electron microscopic studies have failed to detect specific viral particles in the facial nerve [13].

Polymerase chain reaction assays have identified herpes simplex virus in the endoneurial fluid, posterior auricular muscle, and saliva in patients with Bell palsy. [14,15] Shingo Murakami, MD et al analyzed the viral genomes of herpes simplex virus type 1 (HSV-1), varicella-zoster virus, and Epstein-Barr virus using polymerase chain reaction (PCR) on facial nerve endoneurial fluid specimens and specimens of posterior auricular muscle on patients who have undergone decompression surgery. They found HSV-1 DNA in 11 of 14 patients (79%) with Bell palsy and varicella-zoster virus DNA in 8 of the 9 patients (89%) with Ramsay-Hunt syndrome. Hence they conclude Herpes simplex virus type 1 as the major etiologic agent in Bell's palsy[14].

Clinical characteristics of Bell palsy include:

• Peripheral dysfunction of the facial nerve, involving all distal branches

• Abrupt onset (over hours), with maximal facial weakness at 24 to 72 hours

• Unilateral facial weakness that is complete in most patients, but may be partial in as many as one third of patients

• Numbness or pain around the ear on the affected side

• A reduction in taste on the affected side; altered taste on the anterior two thirds of tongue

• Hypersensitivity to sounds (hyperacusis) on the affected side, found in one third of patients

• Spontaneous improvement within 6 months in most cases [16]

• Incomplete healing in 15% of patients, resulting in residual nerve dysfunction, including partial palsy and motor synkinesis (involuntary movement accompanying a voluntary one).

. Also, there should be no signs and symptoms of middle ear disease and posterior fossa disease. There should not be any history of trauma, local infection, tumor, or other central nervous system disease. The diagnosis of idiopathic peripheral facial palsy (PFP) is established by excluding all other possible causes.

Symptoms and signs indicative of possible additional pathology

1. Ear ache, hearing loss

2. Pain or paraesthesia

3. Any abnormality on otoscopy—including otitis media

4. Associated cranial neuropathies or other neurological signs

5. Hypertension

6. Lymphadenopathy, pallor or bruising

7. Vesicles in external meatus or on soft palate

8. Single branch involvement

9. Gradual progression of paralysis beyond 3 weeks

10. Recurrence

11. Mastoid swelling

House-Brackmann facial nerve grading system [17] can be used to quantify the damage.

HOUSE-BRACKMANN---FACIAL NERVE GRADING SYSTEM

Grade	Definition		
	Gross	Rest	Motion

Normal -I	Normal	Normal	Normal
Mild -II	Slight weakness on Close inspection	Symmetry +	Fore head –Good movement. Eye-closure with minimal effort. Mouth –Slight asymmetry
Moderate -III	Obvious	Symmetry +	Forehead-mild to moderate movement. Eye-Closure with maximum effort Mouth- slight weakness with maximum effort.
Mod to Severe. -IV	Obvious & Disfiguring	Symmetry +	Forehead-No movement. Eye-Inability to close with maximum effort. Mouth—Asymmetric movement with maximum effort.
Severe - V	Only barely perceptible motion	Asymmetry	Forehead-No movement. Eye-Incomplete closure. Mouth-Only slight movement.
Total -VI	No movement	No movement	No movement.

DIAGNOSIS

The history and physical examination provide the information which is a key to the diagnosis of Bell palsy. Most patients do not require any laboratory

testing or imaging studies. However, patients who have persistent weakness without significant improvement, involvement of other cranial nerves, or a second episode of palsy require further investigations.

IMAGING

Computed tomography (CT) or MRI is indicated in the following cases:

- No improvement in facial paresis after 1 month
- Hearing loss
- Multiple cranial nerve deficits

MRI with gadolinium is the test of choice

If patient has signs of limb paresis or sensory loss to rule out

Cerebellopontine angle tumor,

Stroke,

Multiple sclerosis, or other structural lesions.

CT scan brain is recommended if a temporal bone fracture is suspected.

ENT evaluation

If hearing loss is suspected, then audiometry testing can be performed to substantiate hearing loss and also help to rule out acoustic neuroma.

Laboratory evaluation.

Laboratory testing is necessary if the patient has signs of systemic involvement such as fever, weight loss, rash, or progressive facial weakness without significant improvement for more than 4 weeks. Number of tests may be helpful:

Complete blood count helps to rule out lymphoreticular malignancy, the first manifestation of which may be peripheral facial palsy. [18]

Blood glucose should be measured if diabetes mellitus is suspected.

Serum antibodies against herpes zoster and Borrelia burgdorferi (the causative agent of Lyme disease) can be checked if the patient has signs such as vesicular lesions on the external ear or if the patient lives in an area where Lyme disease is endemic.

Serum calcium and angiotensin-converting enzyme levels should be tested if sarcoidosis is suspected.

Cerebrospinal fluid testing is helpful if infection or malignancy is suspected; however, cerebrospinal fluid taken from patients with Bell's palsy tends to show mild and inconsistently elevated cell counts and protein levels, but

is otherwise not helpful in identifying the cause.

Electrodiagnostic testing is not routinely done in Bell palsy. It is not very reliable when Bell's palsy is in the initial stages. However, after 2 weeks, it may detect denervation and demonstrate nerve regeneration.

However, in order to prevent irreversible axonal damage, a diagnosis and an early prognostication of the course of the illness are necessary. The pathological process that causes muscle palsy attains a peak during the first two days of illness, but it can have progressive course for 7-10 days, too. Because of these potentially dynamic damages, each prognostic procedure based on clinical manifestation in the early stage of illness is limited. Nevertheless, the lack of any movement of mimic musculature during the first four weeks of illness suggests a bad prognosis [3,16]. Electrophysiological tests may offer valuable information in defining the severity of nerve injury and possible subsequent dysfunction. For these reasons, these tests could be significant prognostic parameters. Previous electrophysiological investigations point to a special prognostic value of the amplitude of Compound muscle action potential (CMAP) in Bell's palsy, since the CMAP amplitude depends on the number of excitable axons. As the nerve fibres degenerate the CMAP amplitude decreases. The degree of degeneration of nerve fibers is directly proportional to the decrease in the CMAP amplitude [19, 20, 21, 22]. Essen [23] and Fish [21] emphasized the decrease in the CMAP amplitude of more than 90%, compared to the healthy side, as a bad prognostic sign. This decrease in the CMAP amplitude is cited in literature as the main criterion for the decision of a

surgical decompression. The diagnostic and prognostic value of the CMAP latency was discussed and disputed in previous studies. Since the CMAP latency reflects the function of the fastest axons, it can remain within a normal range for a long time. Although the amplitude and the latency of CMAP provide valuable information on the distal parts of the nerve damage rate, we cannot estimate the conductivity of the intracranial segment of the nerve using these electrophysiological parameters. Consequently, Blink Reflex plays an important role in the evaluation of the proximal segments of the seventh cranial nerve function and in detection of the intracranial conduction block as the main electrophysiological substratum in Bell's palsy [19, 24]. Therefore, it is a significant prognostic parameter. Electromyography (EMG) investigation of the muscles allows for the registration of action potentials of motor units, as well as spontaneous and insertional activity of the muscles. It is understood that total absence of action potentials during voluntary contractions is a bad prognostic sign [2, 4, 25, 26, 27, 28].

Treatment

Treatment of Bell’s palsy is still controversial. Therapy is difficult to evaluate, because as many as two thirds of patients with Bell’s palsy spontaneously recover and achieve near-normal function. Many patients begin to

improve as early as 10 days after the onset, even without treatment.

Older patients are less likely to recover completely. In a series of 250 patients at an ear, nose, and throat clinic in Greece, the percentage of patients who recovered completely varied from 74% to 83% in patients aged between 4 and 50, whereas the percentage was less than 54% at age 80 and above. [29]

Protection of the eyes

To prevent exposure keratitis, artificial tears should be used frequently during the day in the affected eye to keep it moist. Sunglasses should be worn. Areas with air, contaminated by excessive particulate matter or noxious fumes (construction areas, textile factories) should be avoided. To protect the cornea during sleep, an ophthalmic ointment should be used along with an eye patch. Any corneal abrasion or infection should be treated immediately to avoid possible visual complications. Ophthalmologic consultation is recommended if any deterioration in visual function is noted.

Drug therapy

Drug therapy mainly consists of corticosteroids with or without an antiviral drug (acyclovir). These drugs hasten the recovery and lessen the ultimate degree

of dysfunction. [30, 31, 32] If the patient presents 10 days after the onset of symptoms, no drug treatment is necessary.

Corticosteroids.

A study of 239 patients showed improved rates of synkinesis (involuntary movement accompanying a voluntary one) after treatment with prednisolone 60 mg/day for 10 days and then tapered over 1 week, compared with placebo[33]. The most commonly reported regimen was 1 mg/kg of oral prednisolone, up to 70 mg per day, split into twice-daily dosing. The starting dose was continued for 6 days, and then tapered off over a subsequent 4 days. Corticosteroid therapy reduces autonomic synkinesis and possibly improves the rate of recovery in adults. No study showed significantly worse facial functional outcomes in patients treated with steroids. Four out of five studies done demonstrated a trend for better outcomes in the steroid treated patients.

Corticosteroids plus Acyclovir.

Treatment with antivirals seems logical in Bell's palsy because of the probable involvement of herpes viruses. Acyclovir, a nucleotide analogue, interferes with herpes virus DNA polymerase and inhibits DNA replication. Because of acyclovir's relatively poor bioavailability (15% to 30%), 18 newer drugs in its class are undergoing trials. Better bioavailability, dosing regimens, and

clinical effectiveness in treating shingles have been shown with valaciclovir (prodrug of acyclovir), famciclovir (prodrug of penciclovir), and sorivudine.

Adding acyclovir to prednisolone therapy may improve recovery rates compared with prednisolone alone. A practice parameter from the American Academy of Neurology states that corticosteroids are safe and probably effective, and that acyclovir is safe and possibly effective.[34] Early treatment (i.e., within 3 days after the onset of Bell palsy) is necessary for acyclovir prednisolone therapy to be effective[35] . However, drug therapy may also be effective if started with in 10 days of symptom onset. Adour et al[32] found that 92% of patients regained normal facial motion when given a 10-day course of prednisolone (60 mg a day orally for 5 days, tapered by 10 mg a day for 5 days) and acyclovir (200 mg by mouth five times daily).

The outcome of patients treated with prednisolone plus acyclovir was superior to the outcome of those treated with prednisolone plus placebo, and prednisolone plus acyclovir was "statistically more effective in producing return of volitional muscle motion.[32] Adour et al suggest that if a patient presents within 1 week of the onset of facial weakness and if he or she is not a diabetic or pregnant and has no signs of infection, should receive prednisolone (60 mg/day for 5 days and then a slow taper over 5 days) plus acyclovir (200 mg five times per day for 10 days). Oral valaciclovir, which is converted in the body to acyclovir, has greater bioavailability than oral acyclovir and yields similar acyclovir plasma concentrations with only twice-daily dosing [36]

Corticosteroids Vs Acyclovir.

Acyclovir has been compared with prednisolone. A study comparing prednisolone vs acyclovir found that patients treated with prednisolone had better and complete recovery rates, 93.6% vs 77.7%.[37] Using acyclovir (or any antiviral) without steroids is not advised unless steroids are contraindicated

Further study needed

A larger multicenter double-blind placebo-controlled trial in the future may shed more light on the efficacy of steroids and antiviral therapy.

Surgical treatment

There is no enough evidence to recommend surgical decompression of the facial nerve to hasten the recovery in patients with Bell palsy. Surgical decompression for indications other than trauma is controversial [31, 38]. However, some surgeons still offer middle fossa decompression if electromyography shows a 90% reduction in compound muscle action potential in patients with facial weakness lasting less than 3 weeks [39]. For a patient with permanent facial paralysis despite medical and surgical treatment, many surgical options are available to improve facial function and appearance. These include static sling procedures with

facia lata or alloplastic strips; dynamic procedures with transposition of the temporalis or masseter muscle; hypoglossal-facial nerve anastomosis; crossfacial nerve grafting; free muscle grafting; and microvascular free nerve muscle grafting.

Persistent facial weakness has considerable functional and cosmetic implications. Disfigurement can lead to significant psychosocial morbidity. The small numbers of patients falling into this group require expert assessment. Feedback training and exercise programmes have been shown to provide some benefit in patients with long standing facial nerve paralysis. Surgical techniques aimed at improving function and cosmetics; include nerve repair, graft, or transposition. Nerve transposition involves attaching the distal end of the affected facial nerve to another afferent cranial nerve trunk, for example, the contralateral facial nerve, or a hypoglossal “jump graft”. This technique must be undertaken within two years of paralysis. Muscle transposition, or microneurovascular free muscle transfer, can also be considered.

In summary, patients presenting with acute lower motor neuron facial paralysis require a thorough physical examination. Full neurological examination, otoscopy, and blood pressure measurement are mandatory. In the absence of any abnormal symptoms or signs, further investigation is unnecessary. To date there is no clear evidence that any form of treatment improves outcome of idiopathic facial palsy. Protection of the cornea, with artificial tears and overnight patching, is normally all that is required. Follow up is advisable

Electrotherapy

The use of electrotherapy in the treatment of Bell palsy remains controversial.

Facial exercise

Facial exercises can be beneficial in patients with Bell palsy. They should be performed while standing in front of a mirror and include trying to raise the eyebrows, opening and closing the eyes, blowing, and whistling. These exercises can be performed a few times daily.[40,41] The efficacy of exercise has not been formally evaluated.

Botulinum toxin injections

Motor synkinesis can occur during recovery from Bell's palsy, due to aberrant regeneration of the facial nerve. It can be grossly deforming. Injection of botulinum toxin into the involved muscles is an effective treatment. Hemifacial spasm occasionally appears after Bell's palsy, and blepharospasm can be seen in rare cases.[42.] Both can be treated with injections of botulinum toxin. The time from the resolution of Bell's palsy to the subsequent development of blepharospasm ranges from a few weeks to 24 years.[42.] Hyperlacrimation secondary to aberrant regeneration of the seventh nerve has also been reported after Bell's palsy. Botulinum toxin injection has been used in these patients.

AIMS OF THE STUDY

AIMS OF THE STUDY

1) To assess the Prognostic Value of Electrophysiological Tests in management of Bell's palsy

2) To assess the efficacy of Steroids and Acyclovir in management of Bell's palsy.

MATERIALS AND METHODS

MATERIALS AND METHODS

This prospective study was done on 101 patients with clinical signs of Bell's palsy, of both sexes, in various age groups who attended the Neurology Out patient department, Institute of Neurology. Madras Medical College and Government General Hospital, Chennai, from 01-06-2005 to 01-03-2006. All the patients underwent Neurological and ENT evaluation.

Inclusion criteria

All the patients with Bell's palsy, without clinical evidence of other cranial nerve damage or central nervous system diseases.

Exclusion criteria

Patients with,

Middle ear disease or posterior cranial fossa disease.

Chronic illness like Diabetes mellitus, Hypertension and

Malignancy

were excluded from the experimental group since they may affect the prognosis of the disease.

Treatment of the patient was decided by the treating neurologist in the out patient department and depending on the treatment they receive the patients included in the study are categorized into 4 groups.

Group 1-Patients who received only physiotherapy (Control)

Group 2- Patients who received steroids and physiotherapy.

Group 3- Patients who received steroids, acyclovir and physiotherapy.

Group 4- Patients who received Acyclovir and physiotherapy. .

In the treatment group dosage of steroids used is 1 mg /kg (45 to 60) mg for 5 days then tapered over next 5 days. Acyclovir is used in dose of 1 gm / day for 10 days[32]. The severity of the facial nerve involvement is assessed with House-Brackmann grading (HB) system [17].

HOUSE-BRACKMANN---FACIAL NERVE GRADING SYSTEM

Grade	Definition		
	Gross	Rest	Motion
Normal -I	Normal	Normal	Normal
Mild -II	Slight weakness on Close inspection	Symmetry +	Fore head –Good movement. Eye-closure with minimal effort. Mouth –Slight asymmetry
Moderate -III	Obvious	Symmetry +	Forehead-mild to moderate movement. Eye-Closure with maximum effort Mouth- slight weak with maximum effort.
Mod to Severe. -IV	Obvious & Disfiguring	Symmetry +	Forehead-No movement. Eye-Inability to close with maximum effort. Mouth—Asymmetric movement with maximum effort.
Severe - V	Only barely perceptible motion	Asymmetry	Forehead-No movement. Eye-Incomplete closure. Mouth-Slight movement.
Total -VI	No movement	No movement	No movement.

Grading was done on first visit and on 14th day, 1st, 2nd , 3rd , 6th , 9th and 12th

months to assess the improvement, response to treatment and complications due to the disease and treatment in the various treatment groups.

Nerve conduction study was performed on the 14th day or on the first visit of the patient to the hospital.

For the CMAP examination, supramaximal stimulation was applied for 0.2 ms duration over the trunk of the facial nerve, using the bipolar stimulating electrode with the anode between the ramus of mandible and the mastoid and the cathode in front of the tragus of the ear. The CMAP was recorded with a plate electrode in the target muscles, orbicularis oculi, and orbicularis oris. The amplitude and the latency of the CMAP were analysed.

The mean amplitudes on the affected side were computed as percentage ratio of the normal amplitudes on the healthy side (taken as 100%). The patients were grouped according to the ratio into three groups [2]:

1) A (30 -100%),
2) B (10-30 %),
3) C (less than 10% or not stimulatable).

The latency of the CMAP was also recorded in the same muscles on both

the healthy and affected side. The corresponding mean value was computed, and the patients were grouped according to the latency recorded into three groups[2].

1) A (not greater than 4 ms),
2) B (More than 4 ms), and group
3) C with no CMAP recorded.

In order to estimate prognostic values, the electrophysiological parameters, of amplitude and latency of the CMAPs were correlated with the duration of clinical recovery.

The study groups of patients were clinically tested in a periodic manner within twelve months at various intervals. Based on the clinical recovery, patients were divided into four groups:

A − with recovery during the first two months,
B − recovery within 2-6 months,
C − recovery within 6-12 months, and
D − incomplete recovery after 12 months.

A functional recovery of the seventh cranial nerve was classified based on House-Brackmann (HB) system [17].According to House-Brackmann grading (HB) system the patients were classified into six groups. For analysis, grading done after

second week was taken, because a certain number of patients had a neurological deficit, which had been changing during the first two weeks of the illness.

Chi Square test is employed to test the difference between an actual sample and another hypothetical or previously established distribution such as that which may be expected due to chance or probability. A probability of .05 or less is considered to be a significant.

Relative rate is used frequently in the statistical analysis of binary outcomes where the outcome of interest has relatively low probability. It is thus often suited to clinical trial data, where it is used to compare the outcomes, in people not receiving the new medical treatment (or receiving a placebo) versus people who are receiving an established (standard of care) treatment. In the present study relative rate and confidence intervals of relative rate were calculated using the programme written by DJR Hutchon.

OBSERVATIONS

OBSERVATIONS

One hundred and one patients with signs of the Bell's palsy were included in this study.

Table 1-Age distribution

Age	Control		Steroid		Steroid + Acyclovir		Acyclovir		Total	
	Nos	%	Nos	%	Nos	%	Nos	%	Nos	%
10-20	3	13	12	30	5	17.2	1	11.1	21	20.79
21-30	5	21.7	11	27.5	7	24.1	1	11.1	24	23.76
31-40	8	34.7	9	22.5	11	37.9	2	22.2	30	29.70
41-50	2	8.6	5	12.5	3	10.3	3	33.3	13	12.8
51-60	4	17.3	2	5.0	2	6.8	2	22.2	10	9.9
61-70	1	4.3	1	2.5	1	3.4	0	0	3	2.9
Total	23		40		29		9		101	

Most of the cases belong to the age group of 31-40. Only 2.9% of patients belonged to the age group of 61-70.

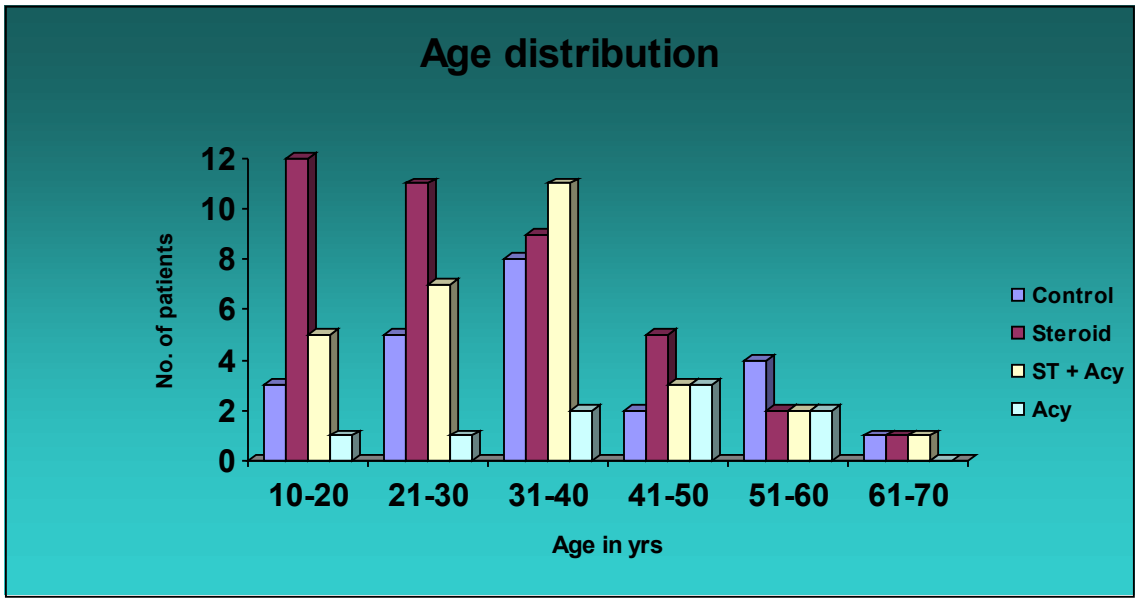


Table 2-Sex distribution

Sex	Control	Steroids	Steroid +Acyclovir	Acyclovir	Total
Male	13	19	16	6	54
Female	10	21	13	3	47
Total	23	40	29	9	101

In the present study sex ratio was almost even. 47 were females (46.6%) and 54 were males (53.4%).

Table 3-Grading

Grade	Control	Steroids	Steroid + Acyclovir	Acyclovir	Total
I	-	-	-	-	-
II	-	-	-	-	-
III	5	7	6	2	20
IV	11	18	13	2	44
V	7	12	08	5	32
VI	0	3	2	-	05
Total	23	40	29	9	101

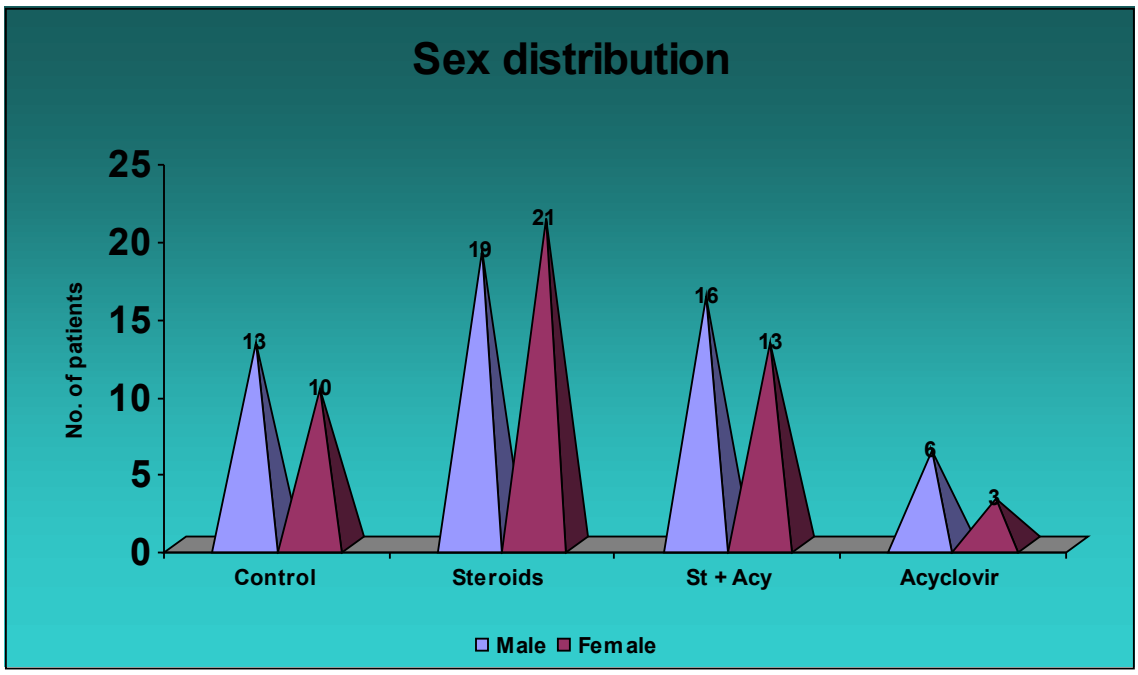
Majority of patients come under grade IV (43.6%) and next comes grade V (31.7%), III (19.8%), and the least in grade VI (4.9%).

Table 4-Analysis of clinical grade and improvement in the study group

Grade	Number of patients	Good improvement	%
III	20	20	100
IV	44	38	86.3

V	32	22	68.8
VI	5	0	0
Total	101	80	79.2

Among the various grades, all the patients in grade showed complete improvement. 86% of percentage of patients improved in grade IV, and 68.8% of patients improved in grade V. None of the patients from grade VI showed improvement.



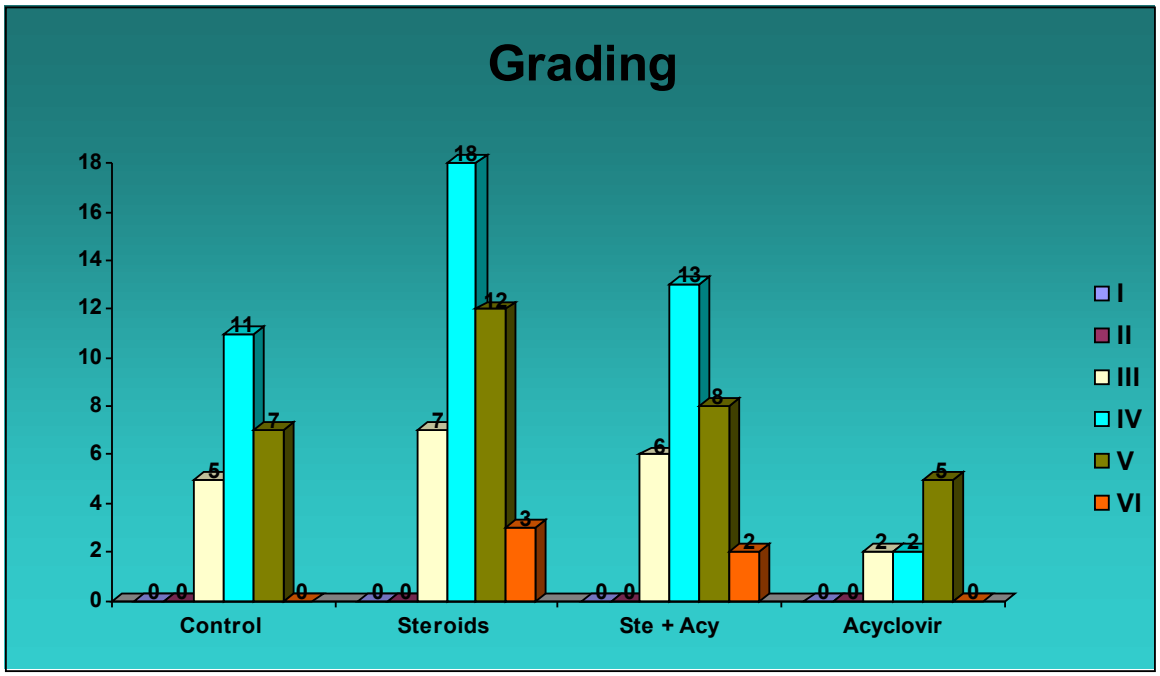


Table 5--Improvement in various age groups-Controls

Age	Grade III		Grade IV		Grade V		Grade VI		Total	
	Nos	Imp	Nos	Imp	Nos	Imp	Nos	Imp	Nos	Imp
10-20	-	-	1	1	2	0	-	-	3	1
21-30	1	1	3	2	1	1	-	-	5	4
31-40	2	2	5	4	1	1	-	-	8	7
41-50	-	-	-	-	2	1	-	-	2	1
51-60	2	2	1	1	1	0	-	-	4	3

61-70	-	-	1	0	-	-	-	-	1	0
Total	5	5	11	8	7	2	-	-	23	15

In the various age groups, one out of three in 10-20 years, four out of five in 21-30 yrs, seven out of eight in 31-40 yrs, one out of two in 41-50 yrs, three out of four in 51-60 yrs and none in 61-70 yrs age groups improved.

Table 6-Improvement in various age groups-Steroids

Age	Grade III		Grade IV		Grade V		Grade VI		Total	
	Nos	Imp	Nos	Imp	Nos	Imp	Nos	Imp	Nos	Imp
10-20	1	1	2	1	7	7	2	0	12	9
21-30	2	2	9	8	1	1	-	-	12	11
31-40	3	3	3	3	2	2	-	-	8	8
41-50	1	1	3	2	1	1	-	-	5	4
51-60	-	-	1	1	1	0	-	-	2	1

60-70	-	-	-	-.	-	-	1	0	1	0
Total	7	7	18	15	12	11	3	0	40	33

In the various age groups, nine out of twelve in 10-20 years, eleven out of twelve in 21-30 yrs, eight out of eight in 31-40 yrs, four out of five in 41-50 yrs, one out of two in 51-60 yrs and none in 61-70 yrs age groups improved.

Table 7-Improvement in various age groups-Steroids + Acyclovir

Age	Grade III		Grade IV		Grade V		Grade VI		Total	
	Nos	Imp	Nos	Imp	Nos	Imp	Nos	Imp	Nos	Imp
10-20	3	3	2	2	-	-	-	-	5	5
21-30	-	-	4	4	3	3	-	-	7	7
31-40	2	2	5	5	3	3	-	-	10	10
41-50	1	1	2	2	1	1	-	-	4	4
51-60	-	-	-	-	-	-	1	0	1	0

61-70	-	-	-	-	1	0	1	0	2	0
Total	6	6	13	13	8	7	2	0	29	26

In the various age groups, all the patients in 10-20 years, 21-30 yrs, 31-40 yrs, 41-50 yrs, improved and none of the patients in 51-60 yrs and 61-70 yrs age groups showed improvement

Table 8-Improvement in various age groups- Acyclovir

Age	Grade III		Grade IV		Grade V		Grade VI		Total	
	Nos	Imp	Nos	Imp	Nos	Imp	Nos	Imp	Nos	Imp
10-20	-	-	-	-	1	1	-	-	1	1
21-30	-	-	-	-	1	1	-	-	1	1
31-40	-	-	1	1	1	0	-	-	2	1
41-50	1	1	-	-.	2	0	-	-	3	1
51-60	1	1	1	1	-	-	-	-	2	2

60-70	-	-	-	-	-	-	-	-	-	-
Total	2	2	2	2	5	2	-	-	9	6

In the various age groups, all the patients in 10-20 years, 21-30 yrs, 51-60 yrs and one out of two in 31-40 yrs, one out of three in 41-50 yrs improved.

Electrophysiology

Table 9- ***Analysis of CMAP recorded in 1st week in 35 patients and recovery***

Amp ratio	Recovery in months			In complete recovery in 12 months	Total
	<2	2-6	>6		
A	15	8	0	3	26
B	0	3	0	5	8
C	0	1	0	0	1
Total	15	12	0	8	35

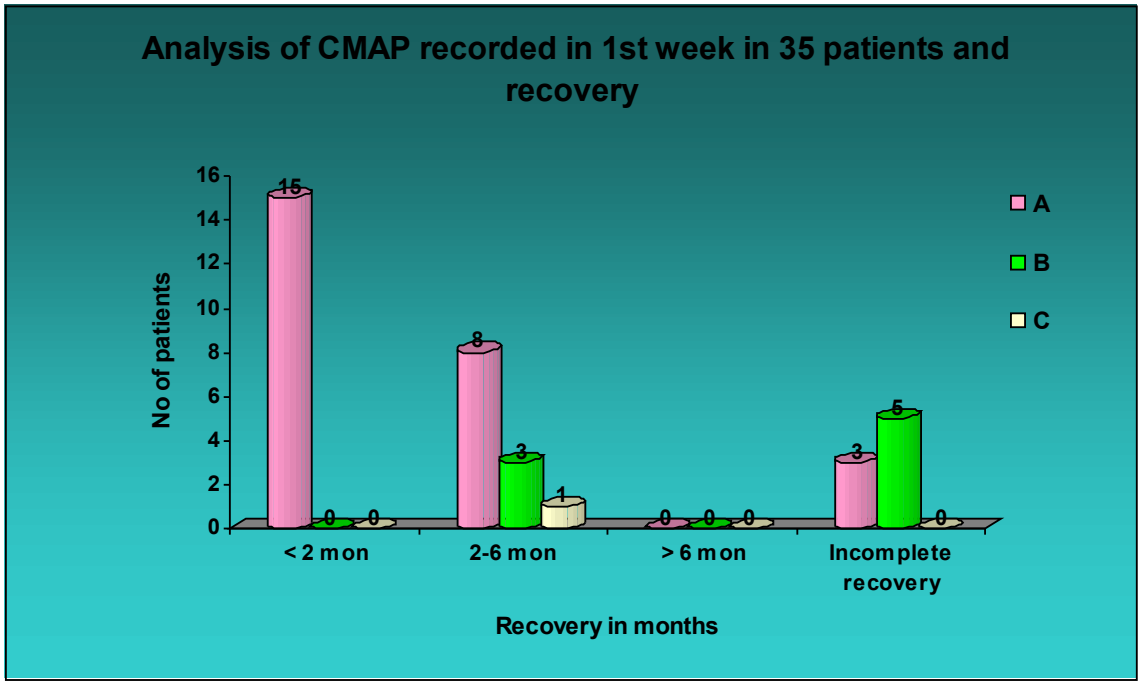
Out of 26 patients in group A, 23 patients had complete recovery. Out of 8 patients in group B, 3 patients recovered, and the one patient in group C had recovered within 6 months

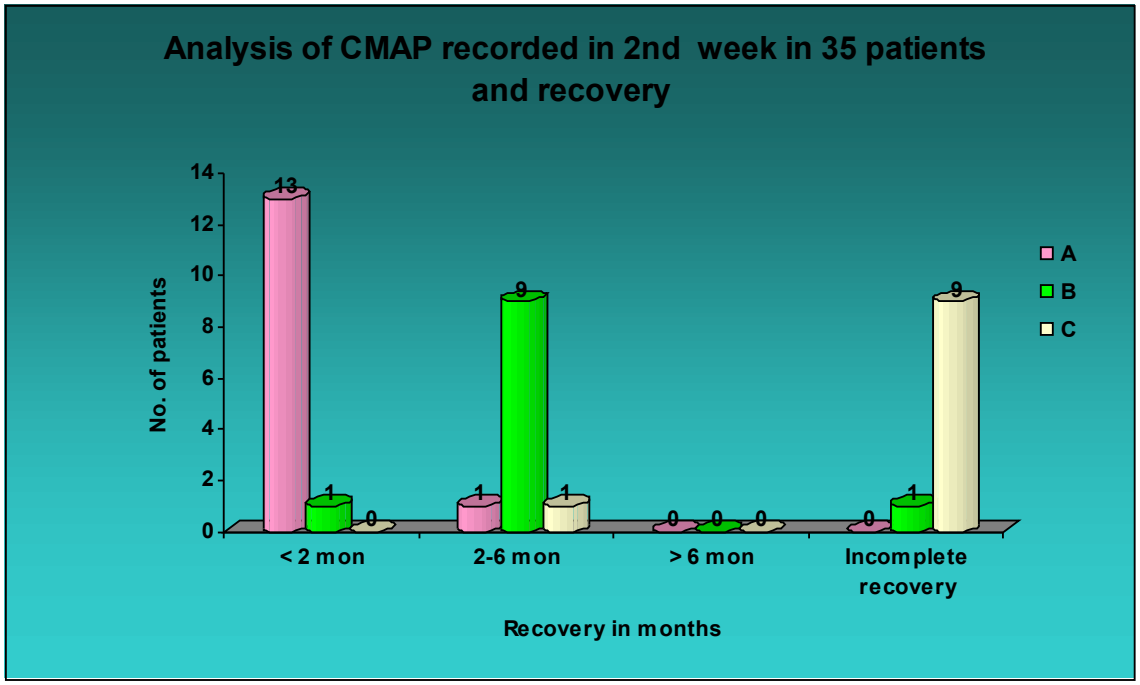
Table 10 - Analysis of CMAP recorded after 2 weeks in same 35 patients and recovery

Amp ratio	Recovery in months			In complete recovery in 12 months	Total
	<2	2-6	>6		

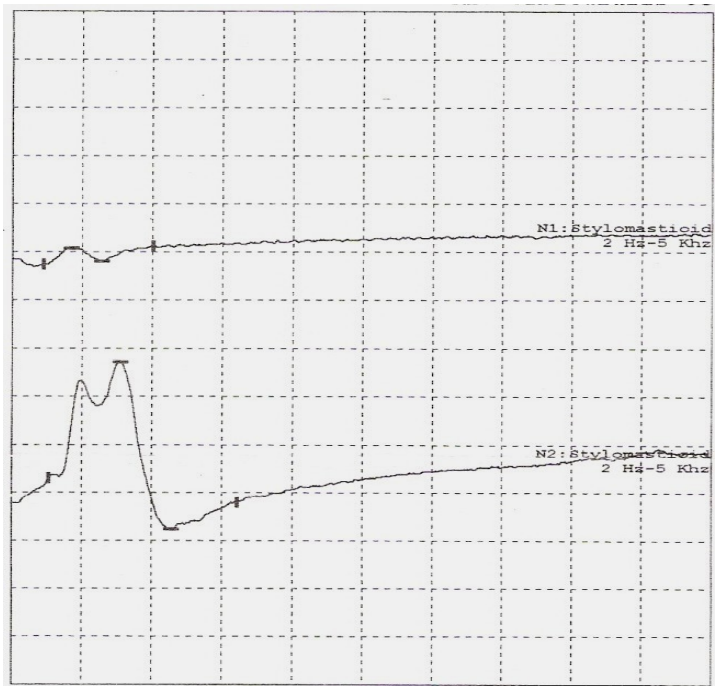
A	13	1	0	0	14
B	1	9	0	1	11
C	0	1	0	9	10
Total	14	11	0	10	35

All the patients in group A had complete recovery. 10 patients showed compete improvement in group B and of 10 cases in group C only one recovered.





CMAP recording showing decreased amplitude in the affected side



CMAP of a patient who had sub clinical involvement on the other side

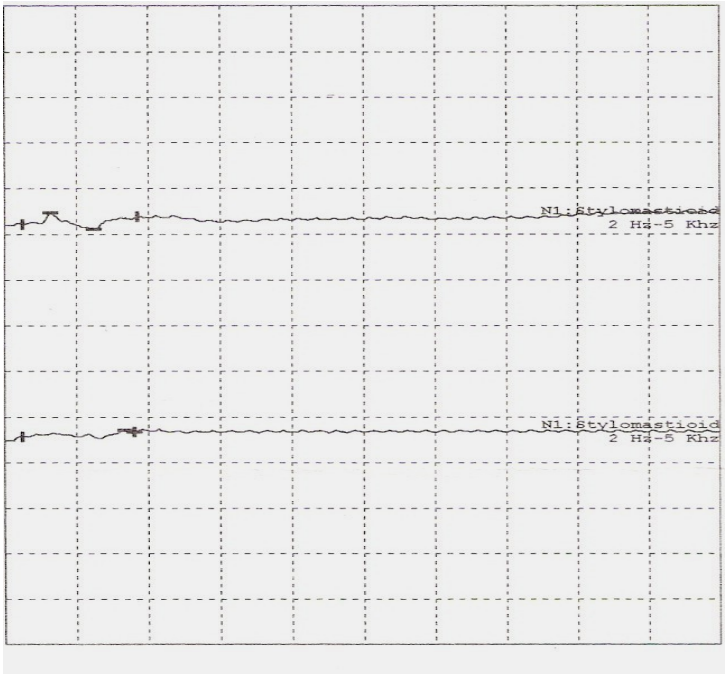


Table 11-*Analysis of recovery in relation to CMAP amp ratio (After 2nd week) in Control group.*

Amp ratio	Recovery in months			In complete recovery in 12 months	Total
	<2	2-6	>6		
A	4	2	0	0	6
B	1	7	1	1	10
C	0	0	0	7	7
Total	5	9	1	8	23

Here all the patients in group A recovered. In group B out of 10 patients 9 recovered and in group C none recovered.

Table 12-Analysis of recovery in relation to CMAP amp ratio (After 2nd week) in Steroid group

Amp ratio	Recovery in months			In complete recovery in 12 months	Total
	<2	2-6	>6		
A	22	1	0	0	23
B	4	4	0	1	9
C	0	1	1	6	8
Total	26	6	1	7	40

Here all the patients in group A recovered within 2 months. In group B out of 9 patients 8 recovered and in group C out of 8 patients 2 recovered.

Table 13-Analysis of recovery in relation to CMAP amp ratio (After 2nd week) in Steroid + Acyclovir group

Amp ratio	Recovery in months			In complete recovery in 12 months	Total
	<2	2-6	>6		
A	16	1	0	0	17
B	3	3	0	1	7
C	0	3	0	2	5
Total	19	7	0	3	29

All the patients in group A recovered within 2 months. In group B out of 7 patients 6 recovered and in group C out of 5 patients 3 recovered

Table 14-*Analysis of recovery in relation to CMAP amp ratio (After 2nd week) in Acyclovir group.*

Amp ratio	Recovery in months			In complete recovery in 12 months	Total
	<2	2-6	>6		
A	2	1	0	0	3
B	0	3	0	0	3
C	0	0	0	3	3
Total	2	4	0	3	9

All the patients in group A and B recovered within 6 months. In group C none of the cases recovered.

CMAP Latency

Table 15- Analysis of CMAP latency recorded in during 1 week and rate of recovery

Latency	Recovery in months			In complete recovery in 12 months	Total
	<2	2-6	>6		
A	17	8	0	10	35
B	0	0	0	0	0
C	0	0	0	0	0
Total	0	0	0	0	35

During the first week the CMAP latency was within normal limits and of the 35 patients 10 had an incomplete recovery

Table 16- Analysis of CMAP latency recorded in after 2 weeks and rate of recovery

Latency	Recovery in months			In complete recovery in 12 months	Total
	<2	2-6	>6		

A	49	24	0	9	82
B	3	2	2	10	17
C	-	-	-	2	2
Total	52	26	2	21	101

82 patients (81.2%) had normal latency, among these, 73 cases recovered within 6 months. Out of 17 who had prolonged latency, 7 patients recovered fully and 2 patients in group C didn't show any improvement.

Rate of improvement in various treatment groups

Table 17-*Control*

Grade	Cases	Good improvement	%
I	-	-	-
II	-	-	-
III	5	5	100
IV	11	8	72.7
V	7	2	28.5
VI	0	0	0
Total	23	15	65.21

All the patients in grade III improved, in grade IV 72.7% improved, and in grade V improvement is 28.5%.

Table 18-*Steroids*

Grade	Cases	Good improvement	%
I	-	-	-
II	-	-	-
III	7	7	100
IV	18	15	83.33
V	12	11	91.66
VI	3	0	0
Total	40	33	82.5

All the patients in grade III improved, in grade IV 83.3% improved, in grade V improvement is 91.6% and none improved in grade VI.

Table 19-Steroids and Acyclovir

Grade	*Cases*	Good improvement	%
I	-	-	-
II	-	-	-
III	6	6	100
IV	13	13	100
V	8	7	87.5
VI	2	0	0
Total	29	26	89.65

All the patients in grade III, IV improved, in grade V 87.5% improved, and none improved in grade VI.

Table 20-Acyclovir

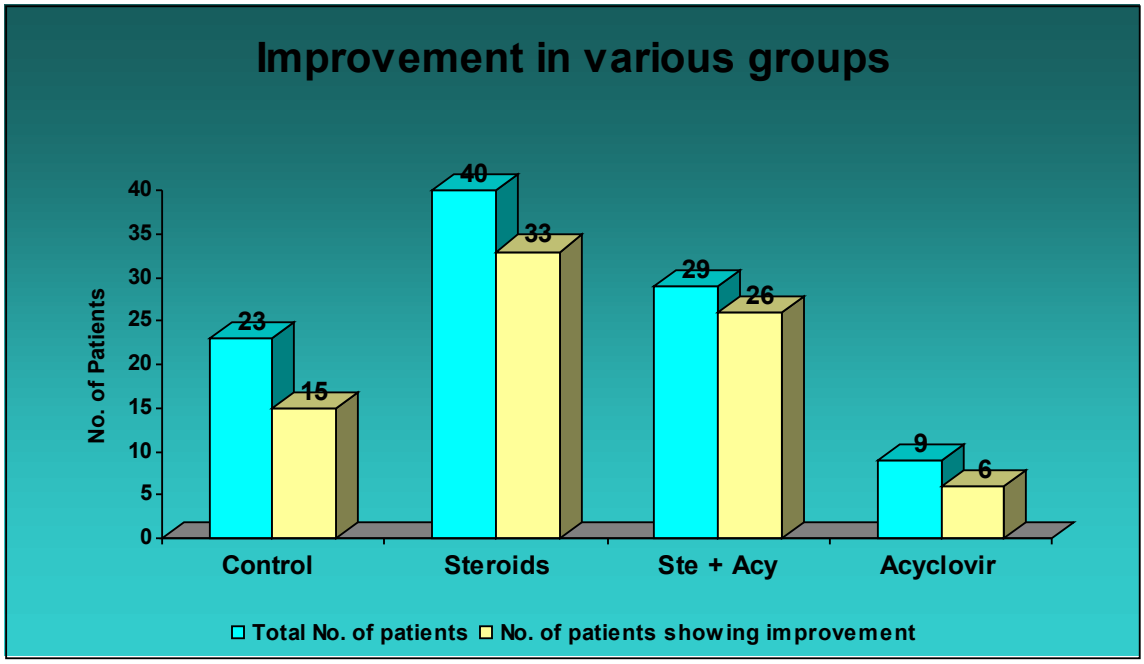
Grade	Cases	Good improvement	%
I	-	-	-
II	-	-	-
III	2	2	100
IV	2	2	100
V	5	2	40
VI	-	-	-
Total	9	6	66.66

All the patients in grade III and IV improved; in grade V improvement is 40%.

Table 21-Rate of improvement in various treatment groups

Groups	Total cases	Patients improved	%
1—Control	23	15	65.2
2—Steroids	40	33	82.5
3—Acyclovir + Steroids	29	26	89.6
4—Acyclovir	9	6	66.6

The improvement in group 1 was 65.2%; group 2 recorded an improvement of 82.5%. Group 3 showed maximum improvement (89.6%) and group 4 the improvement is only 66.6%



Time taken for improvement in various groups

Table 22-Control

Grade	No of cases	Duration for improvement						No of cases improved
		2-4 wks	4-6 wks	6-8 wks	3 months	6 months	12 months	
I	-	-	-	-	-	-	-	-
II	-	-	-	-	-	-	-	-
III	5	-	-	4	1	-	-	5
IV	11	-	-	1	6	1	-	8
V	7	-	-	-	-	1	1	2
VI	-	-	-	-	-	-	-	-
Total	23	-	-	5	7	2	1	15

In the control group the improvement started by 6-8 weeks

Table 23-Steroids

Grade	No of cases	Duration for improvement						No of cases improved
		2-4 wks	4-6 wks	6-8 wks	3 months	6 months	12 months	
I	-	-	-	-	-	-	-	-
II	-	-	-	-	-	-	-	-
III	7	1	4	2	-	-	-	7
IV	18	-	7	7	1	-	-	15
V	12	-	-	5	3	2	1	11
VI	3	-	-	-	-	-	-	0
Total	40	1	11	14	4	2	1	33

Improvement started as early as 2-4 weeks and majority of the patients had complete improvement within 8 weeks.

Table 24-Steroids + Acyclovir

Grade	No of cases	Duration for improvement						No of cases improved
		2-4 wks	4-6 wks	6-8 wks	3 months	6 months	12 months	
I	-	-	-	-	-	-	-	-
II	-	-	-	-	-	-	-	-
III	6	2	4	-	-	-	-	6
IV	13	-	-	8	5	-	-	13
V	8	-	1	4	2	-	-	7
VI	2	-	-	-	-	-	-	-
Total	29	2	5	12	7	-	-	26

Improvement started as early as 2-4 weeks and majority of the patients had complete improvement within 8 weeks. All patients recovered within 6 months.

Table 25-Acyclovir

Grade	No of cases	Duration for improvement						Total cases improved
		2-4 wks	4-6 wks	6-8 wks	3 months	6 months	12 months	
I	-	-	-	-	-	-	-	-
II	-	-	-	-	-	-	-	-
III	2	-	-	2	-	-	-	2
IV	2	-	-	-	2	-	-	2
V	5	-	-	-	2	-	-	2
VI	-	-	-	-	-	-	-	-
Total	9	-	-	2	4	-	-	6

In the above group the improvement started by 6-8 weeks.

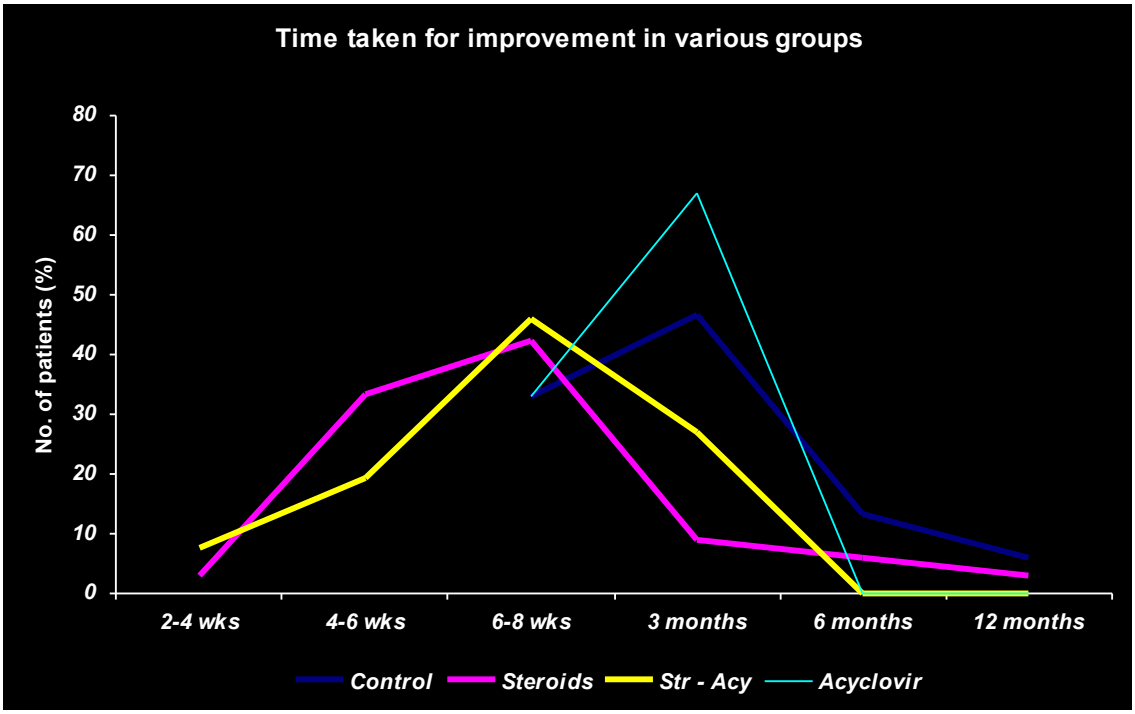


Table 26--Analysis of onset of treatment with improvement in group C (by CMAP ratio)

	Grade IV N =4		Grade V N =4		Grade VI N =5	
	Recovered	Not recovered	Recovered	Not recovered	Recovered	Not recovered
Early treatment	2	0	3	0	0	2
Late treatment	0	2	0	1	0	3

Table 27-Complications

Complications	Control group	Treatment group
Hemifacial spasm	2 (one improved other not improved)	1
Jaw winking	1 (not improved)	0

Table 28-Complications due to steroids

Complications	Group	No. of patients	Age & Sex
Dyspepsia	Steroids	2	65 f & 60 m
Melena	Steroids	1	34 m (Alcoholic)
Vomiting	Steroids + Acyclovir	1	47 f

Table 29-Associated findings

Findings	No. of patients
Antenatal patients	3
Bilateral bell's palsy	1
Recurrent bell's palsy	3

DISCUSSION

DISCUSSION

The study included 101 patients of different age and sex. Sex ratio was almost even (47 women – 46.6%, 54 men – 53.4%). The youngest patient was 11, and the oldest was 65 years old.

The analysis of the age groups showed that the incidence of bells palsy is more common among the younger age group. As the age advances the incidence decreases, with 29.7% in 31-40-age group and 3% in 60-70 age group.

Grading was done in second week of disease and analysis of it shows majority of patients come under grade IV (43.6%) , next comes grade V (31.7%), III (19.8%), and the least in grade VI (4.9%)

One patient had bilateral disease which improved completely with steroids with out any residual deficit. Three patients had recurrence of bell's palsy on same side intervened by an asymptomatic period of six, two and ten years respectively who also completely recovered.

During the study period three antenatal patients were encountered, two with

grade IV weakness and one with grade V weakness. The patients who had grade IV weakness fully improved. The other patient didn't have complete recovery and developed jaw winking.

An outcome of grade I or II using the House and Brackmann facial function scoring system, is considered as good recovery

Age and improvement

Mathews WB et al in his study observed that older age can badly influence the course of the illness [16] .Heath et al [43] presented the results of their research showing that the average age of patients who had a rapid and complete recovery was 35.8 ± 15.9 years, while patients with an incomplete recovery were 55.4 ± 18.8 years old. The results of this study had not shown the existence of a correlation between the age and the duration, degree of clinical recovery. However it is necessary to point out that older individuals had poor recovery when compared to younger individuals. But in these individuals the other factors responsible for poor recovery like, severe degree of weakness, very low CMAP amplitude were present.

Hence the delayed recovery cannot be attributed to age alone; as the other above mentioned factors may also have contributed to the delayed recovery. These results differ from data found in literature, which show that an unsatisfactory

course of the illness can be expected in patients over 45 years of age [16, 43].

Analysis of clinical grade and improvement in the study group

Gordana Djordjević, Stojanka Djurić et al have shown in their clinical research that, in certain number of patients had a changing neurological deficit, during the first two weeks of the illness. They suggesting that the prognosis based on the degree of motor deficit was significantly limited in the early stage of the illness. [44]

The poor correlation between the degree of the paresis in the early stage of the illness, and duration, degree of recovery was observed in the present series also. These observations shows that the degree of motor deficit noted in the first week of the illness was not a clinically reliable parameter in prognosticating the duration of clinical recovery in Bell's palsy. However the clinical grading at 2 weeks when the deficit has stabilized showed correlation with recovery.

It was noted by May M et al and Hauser WA et al [26, 45] that majority of patients with signs of an incomplete facial paralysis of the third and fourth degree, on the fourteenth day of the illness, had a rapid and complete recovery.

The present study shows that patients with signs of an incomplete facial paralysis of third and fourth degree, on the fourteenth day of the illness, had a

rapid and complete recovery. On the other hand, none of the patients in grade VI recovered within 12 months irrespective of the treatment they had received showing that severity of illness was another prognostic factor. These patients on clinical observation showed the sign of a complete paralysis of mimic musculature (VI degree according to H-B) of one half of the face at two weeks of onset. Improvement in grade V was better when compared to grade VI. In this group rate of recovery was affected by the treatment they received and how early it was started.

These results showed that an incomplete facial paralysis had a complete clinical recovery, while a complete paralysis indicated bad prognosis, which is consistent with the literature data. [26,45] An absolute bad prognostic sign was the lack of any movement of the mimic musculature during the first four weeks.

CMAP amplitude

1st week

The results this study showed that progressive decrease of the amplitude was registered from the seventh to the fourteenth day of the illness. In this study out of 101 cases registered, electrophysiological studies were done on thirty five patients in the first week and repeated on the second week. In the other cases electrophysiological studies were done on the fourteenth day of the illness or on the day of first visit to the hospital.

Analysing the CMAP amplitude recorded in the first week and duration of recovery, out of 26 patients in group A, 23 patients had complete recovery and 3 patients did not recover at all. Out of 8 patients in group B, 3 patients recovered and 5 didn't recover and the one patient in group C had complete recovery within 6 months which didn't correlate with observations already made.

Analysing the CMAP recorded in the second week of the illness in the above 35 patients all the patients in group A had complete recovery. Out of 11 patients in group B, 10 patients showed complete improvement and out of 10 cases in group C only one recovered. This observation showed CMAP recorded in second week was very helpful in assessing the prognosis of the disease [44]. This observation correlates with Esslen's reports [23] who showed that the CMAP amplitude decrease is recorded from the third to the tenth day, while Hitoshi et al[46] in their results showed that this decrease occured in the first seven days and remained stable thereafter.

Analysing the CMAP recorded in the second week in this study out of 101 patients 49 patients comes under group A, where all the cases recovered within six months and 44 patients (88%) had complete recovery within two months, remaining 5 cases recovered in 2-6 months among these 5 patients who took more time for recovery, 2 cases belong to control group and each one from other treatment group.

Among the 29 cases under group B, 3 patients didn't have

complete recovery. Among the 26 patients who had complete recovery, 8 patients recovered within 2 months, 17 patients recovered within 2-6 months and only one patient had delayed recovery taking more than 6 months. The observation implies in Group B 86% of cases recovered within 6 months.

Out of 23 patients categorized in Group C, 5 cases recovered completely, of which 4 cases within 6 months and one case took more than 6 months to recover. Above observation makes it clear that if the CMAP amplitude is less than 10 % of the normal side the prognosis is poor

The results observed on the fourteenth day of the illness showed a strong positive correlation between the rate of recovery and CMAP amplitude. The more the amplitude is decreased, the slower was the recovery. Most of the patients whose amplitude was more than 30 % of the normal side recovered completely during the first two months, suggesting mild damage of the nerve (neuropraxia). The patients whose amplitude values were between 10-30% recovered within 6 months which corresponded to the second type of the nerve damage (axonotemesis). The distinct amplitude decrease to (0-10%) pointed to a severe nerve damage (neurotmesis) and an incomplete clinical recovery. The above observation correlated well with the observations made by others. [44]

In this study there was decrease of CMAP amplitude in 7 patients in the

clinically normal side which did not show any motor deficit. Among the seven cases, 5 cases showed this abnormality in the first week and the other two cases on 9th and 11th day respectively depicting sub clinical involvement on the other side also. Since electrophysiological studies were done only in 35 patients in the first week this may be an under estimation. If electrophysiological studies were done in the first week in all cases the identification of the sub clinical cases may be high. Similar observation was made by Natarajan V et al [47] who had documented 20% of sub clinical cases in their study.

CMAP latency.

The role of the CMAP latency in the early diagnosis and prognosis of Bell's palsy is uncertain. Although some researchers showed that abnormal latency can point to a bad prognosis, it is believed that this factor has a limited significance. Since the latency reflects the function of the fastest fibers, it can stay within normal values for a long time even in cases of a distinct axonal loss. Gilliat and Taylor [48] reported that latency stays within normal values until the M potential is lost. However, some studies have demonstrated that abnormal latency can be suggestive of a bad prognosis. Langwort and Taverner [49] emphasized that the extreme prolongation of the CMAP latency or absence of CMAP as a bad prognostic sign. Danielides et al. have provided the results of their study made in 1994 and 1996[50] which showed that the latency extension results in a bad

prognosis of the illness. They also claim that the reliability of this feature was less important than CMAP amplitude for the prognosis.

The results of this study shows that during the first seven days of the illness latency values were normal with or without a minimal asymmetry compared to the healthy part, while after the fourteenth day the prolongation of the CMAP latency was above normal values in some cases . On the fourteenth day after the first symptom the prolongation of the latency was registered in 19 (18.8 %) patients and the latency was within normal limits in the remaining 82 (81.2%) patients.

Analysis of the CMAP latency values and the duration of the recovery during the first week did not show the existence of the correlation. After the fourteenth day a strong positive correlation was registered - longer latency resulted in slower recovery.

Out of 101 patients 82 patients (81.2%) had normal latency (group A), among these, 73 cases recovered within 6 months. The other 9 cases who did not have recovery had a severe decrease in CMAP amplitude.

In the remaining 19 patients, 17 had prolonged latency (group B) and in 2 patients CMAP was not obtained (group C). In this (group B) out of 17 patients

7 patients recovered fully and the remaining 10 patients did not recover at all. The patients in group B who had good recovery the amplitude belonged to group A and B category and the 10 patients who didn't recover had a grossly reduced CMAP amplitude.

The 2 patients in group C didn't show any improvement. It was observed poor recovery is a rule if latency along with CMAP amplitude is reduced.

This observation showed that even though latency measurement as an independent factor is not much helpful in assessing the prognosis, if combined with the amplitude predicts the prognosis.

Treatment

During the study, four groups were formulated.

Group 1-Patients who received only physiotherapy (Control)

Group 2- Patients who received steroids and physiotherapy.

Group 3- Patients who received steroids, acyclovir and physiotherapy.

Group 4- Patients who received Acyclovir and physiotherapy. .

In this study 23cases were enrolled in group 1, 40 patients in group 2, 29 patients group 3, and 9 group 4. The age and sex, clinical grade in each group were comparable.

Rate of recovery

For assessing the therapeutic effect in the various groups the proportion of patients recovering good facial function in the treated group were compared with the proportion of patients recovering good facial function in the control group. Relative rate (RR) was calculated by means of the following formula [34]

Relative rate = [A / (A+C)] / [B / (B+D)]

A = Number of patients with good recovery in treatment group
B = Number of patients with good recovery in control group
C = Number of patients with poor recovery in treatment group
D = Number of patients with poor recovery in control group

95% confidence interval was also calculated for the relative rate.

Relative rate--- Steroids vs Control ==1.265 (95% CI 0.90-1.76)

Relative rate--- Steroids +Acyclovir vs Control ==1.374 (95% CI 0.99-1.89)

Relative rate--- Steroids +Acyclovir vs Steroids ==1.086 (95% CI 0.89-1.31)

Relative rate--- Acyclovir vs Control ==1.022 (95% CI 0.58-1.77)

In this study, the best functional recovery was seen in patients who were treated with steroids and Acyclovir. The above data was statistically significant when compared to control group with 'P' value of 0.031668.

Patients treated with steroids alone also had a better recovery when compared with control group. The data interpretation showed that, the steroid group scored over the control group by 1.26 times. However it was not statistically significant.

Patients treated with steroids and Acyclovir scored over patients treated with steroids by 1.08 times. Patients treated with Acyclovir alone scored over control by 1.02 times. The above data was not statistically significant.

These observations correlated with other studies[51,52] which says that steroids hasten clinical recovery in bell's palsy . Wolf et al have suggested that patients with complete facial palsy benefit most from steroids.[53]

Combination of Steroids and acyclovir is definitely useful when compared to the control group. The combination scores over the control group by 1.37 times which is statistically significant (P value= 0.031668). This observation correlated with the study made by Adour and colleagues.[32] The present study did not find any beneficial effect in treating the patients with Acyclovir alone. Even though the study sample is small this observation concurs with the observation made by De Diego JI, et all in the study comparing prednisolone vs acyclovir. They found that patients treated with prednisolone had better complete recovery rates, 93.6% vs 77.7%. [37]

The observations made in the present study shows that the combination of steroid and acyclovir is definitely useful in Bell's palsy. Steroids are probably effective in management of bells palsy as they improve the rate of recovery. Out come in patients in whom Acyclovir alone (group 4) was used didn't score over

the control group.

Time taken for recovery

Analysing the time taken for improvement in the various groups, the recovery in control group started by 6-8 weeks while in treatment group improvement started between early by 2-4 weeks. Moreover on comparing the recovery of patients in grade IV, with in control group only 12.5 % improved within 2 months, while in treatment group more than 60 % improved within 2 months. The above observation implied recovery starts early in treatment group when compared with control group. Taverner et al in his study [54], reported a trend for steroid-treated patients to recover faster than the control patients, but the differences were not significant

.

Role of early treatment in bells palsy.

Williamson IG et al and May MM et al have suggested that steroids work best in patients with Bell's palsy if started early.[3,55] Shafshak however defers this

observation in his study[52]. Analysing the outcome of patients in group C, (by CMAP ratio) those who were treated, five out of thirteen, had good recovery. Among the patients who recovered, two patients had grade IV deficit and two had

grade V deficit, and all the cases received treatment within 3 days of the illness. In the eight patients who had not recovered fully, five patients had complete palsy (grade VI) and treatment was started late in three cases. In the other three patients treatment was started late and they belonged to grade IV and V. The above observation implies that early treatment in cases with incomplete paralysis hastens the recovery and in patients with complete paralysis there is no impact of early treatment.

Analysing the complications and synkinesis, hemi facial spasm occurred in 2 cases in control group and in one patient in the treatment group. Synkinesis occurred in one case in control group this correlated with other author's observation. But review by quality standard subcommittee of the American academy of neurology concluded that, none of the studies described a significant decrease in the frequency of autonomic synkinesis (e.g., "crocodile tears") in patients treated with steroids [34]

Complications due to treatment.

Three studies [54, 56, 57] discussed steroid side effects. Side effects occurred in 1 to 4% of treated patients. These side effects, in descending order of frequency,

are dyspepsia, loss of blood sugar control, recurrent duodenal ulcers, mood swings, and acute psychosis. The present study observed treatment related complications in four patients.

Two patients who developed dyspepsia were more than 65 years old. The patient who had melena after therapy was also an alcoholic. One 47 year old patient treated with steroids and acyclovir developed vomiting. This observation indicates that steroid related complications are more among the older people and persons who are having other risk conditions. Otherwise treatment of Bells palsy with steroids is effective and safe.

Conclusions

CONCLUSIONS

- The most reliable prognostic data is obtained after the fourteenth day from the onset of palsy.
- Electrophysiological studies can predict duration of the clinical recovery and the outcome of the illness.
- Amplitude ratio of CMAP is the most reliable parameter in assessing the prognosis
- Latency measurement as an independent factor is not much helpful in assessing the prognosis, however when combined with the amplitude ratio predicts the prognosis.
- Simultaneous subclinical facial nerve involvement does occur on the contralateral side in 16% of the cases.
- Bell's palsy patients with incomplete facial paralysis have excellent outcomes
- Steroids are safe and are probably effective in management of bells palsy which improves the rate of recovery.
- Acyclovir in combination with prednisolone is safe and has a definite role in improving facial functional outcomes in patients with Bell's palsy than using the drug alone
- Early treatment in cases with incomplete paralysis hastens the recovery and it has no effect on cases with severe deficit.

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PROFORMA

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INSTITUTE OF NEUROLOGY
MADRAS MEDICAL COLLEGE. CHENNAI
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BELL'S PALSY-PROFORMA

Si no: Min no:

Name Age- Sex Occupation:
Address: Phone no:
E-mail:

Date of onset Duration Side

Symptoms:

Ear pain Deviation of angle of mouth

Difficulty in closing eyes *Other symptoms*

Examination

Forehead wrinkling Orbicularis occuli

Mouth weakness Taste

Hyperacusis Others

Diease grade: Past history

Associated illness: D.M / HT /PT

INVESTIGATIONS

Urine Alb Sug Dep

Blood: Hb Tc Dc ESR:

Urea Sugar Creatinine

Treatment

1) Physiotherapy

2) Steroids + Physiotherapy

3) Steroids + Acyclovir

4) Acyclovir + Physiotherapy

Remarks

Follow-up

1) Clinical

	Onset	1-Month	2-Months	3-Months	6-Months	12-Months
House brachman Grade						

2) Electrophysiological evaluation

Visit	LEFT			RIGHT		
	Latency	Duration	Amplitude	Latency	Duration	Amplitude

3) Complications

	Onset	1-Month	2-Months	3-Months	6-Months	12-Months
Hemi-spasms						
Synkinesis						
Drug related						

HOUSE-BRACKMANN---FACIAL NERVE GRADING SYSTEM

Grade	Definition		
	Gross	Rest	Motion
Normal -I	Normal	Normal	Normal
Mild -II	Slight weakness on Close inspection	Symmetry +	Fore head –Good movement. Eye-closure with minimal effort. Mouth –Slight asymmetry
Moderate -III	Obvious	Symmetry +	Forehead-mild to mod movement. Eye-Closure with maximum effort Mouth- slight weakness with maximum effort.
Mod to Severe. -IV	Obvious & Disfiguring	Symmetry +	Forehead-No movement. Eye-Inability to close with maximum effort. Mouth—Asymmetric movement with maximum effort.
Severe - V	Only barely perceptible motion	Asymmetry	Forehead-No movement. Eye-Incomplete closure. Mouth-Only slight movement.
Total -VI	No movement	No movement	No movement.

MASTER CHART

MASTER CHART

CONTROL-Group

Si No	Age	Sex	R/L	Du	Gra	NCS				Follow-Up Grade						Comp	Drug side effects
						Ratio 1st week		Ratio 2nd week		2-4 Wks	4-6 Wks	6-8 Wks	3 Mon	6 Mon	12 Mon		
						Lat	Amp	Lat	Amp								
1	51	M	R	7	III	A	A	A	A	III	III	II	I	I	I	-	-
2	56	M	R	3	V	A	A	C	C	V	V	IV	IV	IV	IV	HFS	-
3	40	F	L	1	IV	-	-	A	B	IV	III	III	II	I	I	-	-
4	37	M	R	15	V	-	-	A	B	V	IV	IV	III	II	I	-	-
5	31	F	L	13	IV	-	-	A	A	IV	IV	III	II	I	I	-	-
6	30	F	L	16	V	-	-	B	C	V	V	V	IV	IV	IV	-	-
7	24 AN	M	R	5	IV	-	-	B	B	IV	IV	III	III	II	I	-	-
8	28	F	R	11	III	-	-	A	B	III	III	III	II	I	I	-	-
9	41	M	L	50	V	-	-	B	C	V	V	IV	IV	IV	IV	-	-
10	40	M	L	7	IV	A	A	A	A	IV	III	II	I	I	I	-	-
11	54	M	L	7	III	-	-	A	B	III	III	II	I	I	I	-	-
12	37	F	L	11	III	A	A	A	A	III	III	II	I	I	I	-	-
13	18	F	R	14	V	-	-	C	C	V	IV	III	III	III	III	-	-
14	43	M	L	12	V	-	-	B	B	V	IV	III	III	III	II	HFS	-
15	32	M	R	22	IV	-	-	A	C	IV	IV	III	III	III	III	-	-
16	18 AN	F	R	12	V	-	-	B	C	V	V	IV	IV	III	III	JW	-
17	51	M	R	6	IV	A	A	A	B	IV	III	III	II	I	I	-	-
18	31	M	L	14	III	-	-	A	A	III	III	I	I	I	I	-	-
19	16	F	R	15	IV	-	-	A	B	IV	III	III	II	I	I	-	-
20	38 AN	F	L	2	IV	A	C	A	B	IV	III	III	II	I	I	-	-
21	28	M	R	10	IV	-	-	A	B	IV	III	III	III	III	III	-	-
22	61	M	R	17	IV	-	-	B	C	IV	IV	III	III	III	III	-	-
23	30	F	L	11	IV	-	-	A	A	IV	III	III	II	I	I	-	-

STEROIDS-Group

Si No	Age	Sex	R/L	Du	Gra	NCS				Follow-Up Grade						Comp	Drug side effects
						Ratio 1st week		Ratio 2nd week		2-4 Wks	4-6 Wks	6-8 Wks	3 Mon	6 Mon	12 Mon		
						Lat	Amp	Lat	Amp								
1	16	M	R	2	V	A	A	A	A	IV	III	II	I	I	I	-	-
2	37	F	L	7	IV	-	-	A	A	III	III	I	I	I	I	-	-
3	28	M	L	3	IV	A	A	A	A	III	I	I	I	I	I	-	-
4	19	M	R	2	V	A	B	A	B	V	IV	III	II	I	I	-	-
5	65	F	R	2	VI	A	A	B	C	VI	VI	V	IV	III	III	DP	-
6	50	F	L	3	III	-	-	A	A	III	I	I	I	I	I	-	-
7	37	M	L	2	III	A	B	A	B	III	I	I	I	I	I	-	-
8	30	F	L	1	IV	-	-	A	A	IV	III	II	I	I	I	HFS	-
9	19	F	R	6	VI	A	B	A	C	VI	IV	III	III	III	I	-	-
10	25	M	R/L	4	IV	-	-	B	A	IV	II	I	I	I	I	-	-
11	60	M	L	2	IV	-	-	A	A	III	II	I	I	I	I	-	-
12	17	M	L	2	V	-	-	A	A	IV	III	II	I	I	I	-	-
13	19	F	R	3	III	A	A	A	A	III	I	I	I	I	I	-	-
14	22	M	L	4	V	A	A	A	B	IV	III	II	I	I	I	-	-
15	34	M	L	6	V	-	-	A	B	IV	IV	III	II	I	I	Mele	-
16	33	F	R	4	IV	-	-	A	A	III	I	I	I	I	I	-	-
17	17	F	R	7	IV	A	A	B	C	IV	IV	III	III	III	III	-	-
18	40	M	L	4	IV	-	-	A	A	III	II	I	I	I	I	-	-
19	31	M	L	2	III	A	A	A	A	III	I	I	I	I	I	-	-
20	17	F	L	1	VI	A	B	B	C	VI	V	III	III	III	III	-	-
21	18	F	R	5	V	-	-	A	A	IV	III	II	I	I	I	-	-
22	28	F	L	3	IV	A	A	A	B	IV	III	III	II	I	I	-	-
23	30	M	R	3	IV	A	A	A	B	IV	III	II	I	I	I	-	-
24	60	M	L	6	V	A	B	A	C	V	V	V	IV	IV	III	DP	-
25	50	F	R	4	IV	-	-	A	A	III	II	I	I	I	I	-	-
26	37	F	L	3	III	-	-	A	A	III	III	II	I	I	I	-	-
27	26	F	R	4	IV	-	-	A	A	III	I	I	I	I	I	-	-

28	30	M	L	7	IV	A	A	A	C	IV	IV	III	III	III	III	-	-
29	18	F	R	5	V	-	-	A	A	V	IV	II	I	I	I	-	-
30	50	M	L	10	IV	-	-	A	B	IV	IV	III	III	III	III	-	-
31	30	F	R	1	III	A	A	A	A	II	I	I	I	I	I	-	-
32	47	M	L	1	V	A	A	A	C	IV	IV	III	III	II	I	-	-
33	17	M	R	2	V	-	-	B	C	V	IV	III	III	III	II	-	-
34	34	F	R	9	V	-	-	A	B	V	IV	III	III	II	I	-	-
35	28	F	L	2	III	-	-	A	A	III	II	I	I	I	I	-	-
36	13	M	R	1	V	-	-	A	A	V	IV	III	I	I	I	-	-
37	44	F	R	6	IV	A	A	A	B	IV	III	II	I	I	I	-	-
38	30	F	R	4	IV	-	-	A	A	IV	III	II	I	I	I	-	-
39	26	M	L	1	IV	-	-	A	A	IV	III	I	I	II	I	-	-
40	19	F	R	2	IV	-	-	A	A	IV	III	II	I	I	I	-	-

STEROIDS + ACYCLOVIR-Group

Si No	Age	Sex	R/L	Du	Gra	NCS				Follow-Up Grade						Comp	Drug side effects
						Ratio 1st week		Ratio 2nd week		2-4 Wks	4-6 Wks	6-8 Wks	3 Mon	6 Mon	12 Mon		
						Lat	Amp	Lat	Amp								
1	33	M	R	4	IV	-	-	B	B	IV	III	II	I	I	I	-	-
2	38	F	L	8	V	-	-	A	B	IV	III	II	I	I	I	-	-
3	51	M	L	7	VI	A	B	B	C	VI	VI	V	V	IV	IV	-	-
4	47	F	R	1	III	A	A	A	A	III	I	I	I	I	I	-	Vom
5	46	F	L	5	IV	-	-	A	B	IV	IV	III	II	I	I	-	-
6	28	M	L	3	V	-	-	A	C	IV	III	III	II	I	I	-	-
7	18	M	R	1	III	A	A	A	A	II	I	I	I	I	I	-	-
8	30	F	L	2	IV	-	-	A	A	III	III	I	I	I	I	-	-
9	41	M	L	10	V	A	A	A	A	IV	III	II	I	I	I	-	-
10	37	M	R	1	IV	-	-	A	B	III	III	I	I	I	I	-	-
11	36	F	L	2	V	A	A	A	A	IV	III	II	I	I	I	-	-
12	28	F	R	7	V	-	-	A	A	III	III	II	I	I	I	-	-
13	31	M	L	9	IV	A	B	A	B	IV	III	III	II	I	I	-	-
14	18	M	R	2	III	-	-	A	A	I	I	I	I	I	I	-	-
15	17	F	R	5	IV	-	-	A	A	III	III	II	I	I	I	-	-
16	36	M	L	2	III	-	-	A	A	III	II	I	I	I	I	-	-
17	40	M	R	1	IV	-	-	A	A	IV	III	I	I	I	I	-	-
18	37	F	L	3	IV	-	-	A	B	III	III	II	I	I	I	-	-
19	36	F	R	7	V	A	A	A	B	V	IV	III	II	I	I	-	-
20	28	M	R	3	IV	A	A	A	A	IV	III	III	II	I	I	-	-
21	21	M	R	2	IV	-	-	A	A	IV	III	I	I	I	I	-	-
22	20	M	L	2	III	-	-	B	A	III	I	I	I	I	I	-	-
23	29	F	R	1	V	A	A	A	A	IV	II	I	I	I	I	-	-
24	40	M	L	3	III	-	-	A	A	III	II	I	I	I	I	-	-
25	61	M	R	8	V	A	A	A	B	V	IV	III	III	III	III	-	-
26	60	M	L	7	VI	A	B	A	C	VI	VI	V	IV	IV	IV	-	-
27	38	F	R	2	IV	-	-	A	C	IV	III	III	II	I	I	-	-
28	18	F	L	2	IV	-	-	A	A	IV	III	II	I	I	I	-	-
29	21	F	R	2	IV	-	-	A	C	IV	III	III	II	I	I	-	-

ACYCLOVIR--Group

Si No	Age	Sex	R/L	Du	Gra	NCS				Follow-Up Grade						Comp	Drug side effects
						Ratio 1st week		Ratio 2nd week		2-4 Wks	4-6 Wks	6-8 Wks	3 Mon	6 Mon	12 Mon		
						Lat	Amp	Lat	Amp								
1	51	M	R	15	III	-	-	A	A	III	III	I	I	I	I	-	-
2	48	M	L	11	V	-	-	B	C	V	V	IV	IV	III	III	-	-
3	47	F	R	12	III	-	-	A	A	III	III	II	I	I	I	-	-
4	38	M	L	4	V	-	-	A	C	V	V	V	V	V	V	-	-
5	34	M	L	3	IV	-	-	A	B	IV	III	III	II	I	I	-	-
6	57	F	R	14	IV	-	-	A	A	IV	III	III	II	I	I	-	-
7	43	M	L	18	V	-	-	A	C	V	V	IV	III	III	III	-	-
8	30	M	R	20	V	-	-	B	B	V	III	III	II	I	I	-	-
9	11	F	L	18	V	-	-	A	B	V	IV	III	II	I	I	-	-
10																	

Key to master chart

R / L --Side affected, Right or Left

Du ---Duration of illness in days

Lat --Latency of CMAP

Amp ---Amplitude ratio of CMAP

Wks -- Weeks

Mon ---Months

F -- Female

M ---Male

Comp -- Complications

DP ---Dyspepsia

Mele -- Melena

HFS ---Hemi facial spasm

JW --Jaw winking

Vom ---Vomiting

A, B, C --Group as per CMAP, Latency, Amplitude ratio

I, II, III, }
IV, V, VI } --Grade as per based on House-Brackmann (HB) system